Carriers of meningococci among staff from department in which meningococcal disease was present and from control departments

	No of staff	No (%) of carriers	Groups of meningococci		
			C	B	Other
Haematology department:					
Bedside contact with index case	68	11 (16)	5	3	3
No contact	115	9 (8)	0	4	5
Infectious diseases and rheumatology departments	247	20 (8)	0	10	10
Total	430	40 (9)	5	17	18

were taken from all carriers three days after the end of treatment with rifampicin showed that meningococci were no longer carried.

Comment

Strains of meningococci carried in the general population usually belong to non-virulent serological groups and types, even during an epidemic, and hence the ratio of attacks to carriers is low[3]; in Denmark in 1987 it was 1:17 000 (297 cases in a population of 5·1 million). If the secondary attack rate among household contacts of an index case of meningococcal disease is 500-800 times that in the general population[4] then the estimated risk is 2·9-4·8%. Three of seven carriers identified in our study developed the disease, giving an attack rate of 43% (95% confidence interval 7% to 82%); consequently we gave chemoprophylaxis to household contacts of carriers.

Patients with lower respiratory tract infection from whom *N meningitidis* is isolated most commonly suffer from chronic pulmonary disease.[5] The relative risk of secondary meningococcal disease among people in their vicinity is unknown. We therefore suggest that until more evidence has accumulated chemoprophylaxis should be considered for patients in the same room as a patient with respiratory tract infection caused by virulent meningococci of serogroup C:2a and for staff in contact with such patients.

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Does eradication of meningococcal carriage in household contacts prevent secondary cases of meningococcal disease?

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From 1 October 1981 to 31 March 1988, 109 cases of meningococcal disease, mainly due to group B type 15 subtype 16 strains resistant to sulphonamide, were recorded in Gloucester Health District, six of them occurring among 309 household contacts of index patients. Two cases occurred 12 and 36 hours after admission of the index patient and before chemoprophylaxis was given. The four others occurred 28, 107, 147, and 156 days after the index cases. All household contacts and three of the four index patients had received rifampicin for two days as currently recommended,[1] and postnasal swabs were negative after treatment.

Effectiveness in eradicating nasopharyngeal carriage of meningococci is considered an appropriate criterion for selecting chemoprophylactic agents for meningococcal disease.[2] As most previous studies have examined short term effectiveness[3] and as four secondary cases in this outbreak occurred up to five months after prophylaxis we examined whether carriage of outbreak strains was persistently reduced after rifampicin was given.

Subjects, methods, and results

During a community survey of 6234 people in November 1986,[4] 79 nasopharyngeal carriers of outbreak strains were identified. In December after a second postnasal swab 50 carriers received rifampicin (600 mg twice daily for adults, 10 mg/kg twice daily for children) for two days, and 29 declined treatment. Thirty three (66%) in the treated and 18 (62%) in the untreated groups were still carrying outbreak strains (table).

In January 1987 only one of the treated group had a positive postnasal swab compared with 13 of the untreated group (table). If the natural rate of loss (28%) had applied to the treated group then the expected number of carriers would have been 24. Thus the effectiveness of rifampicin in eradicating carriage was 96% (23/24, 95% confidence interval 88 to 100%). In May and November two more patients in the treated group had positive postnasal swabs, and rates of carriage continued to fall slowly in the untreated group.

Numbers (percentages) of people with nasopharyngeal swabs positive for outbreak strains of meningococci among treated and untreated carriers

Treatment	Time when swab taken			
	At time of treatment (December 1986)	After one month	After five months	After 11 months
Rifampicin	33/33 (100)	1*/33 (3)	2/32 (6)	2/30 (7)
None	18/18 (100)	13/18 (72)	10/17 (59)	5/15 (33)

*Patient received second course of rifampicin and swab was subsequently negative.

Comment

Support for the practice of prescribing antibiotics such as rifampicin to household contacts of patients with meningococcal disease[1,2] comes from a retrospective study of rates of secondary attack with limited follow up.[2] Unlike the policy of mass prophylaxis in military communities,[3] it has never been evaluated by controlled trial.

Our study showed that persistent eradication of nasopharyngeal carriage of meningococci can be achieved by giving rifampicin for two days. Despite the apparently low rate of reacquisition four cases occurred one to five months after prophylaxis and the rate of

secondary attack was higher than expected.[2 5] Household contacts of a patient had a relative risk of infection 750 times that of other people in the health district (1·94/0·0026). Possibly the rate of secondary attack would have been higher still without prophylaxis. Possibly, too, prophylaxis did not reduce rates of attack but merely delayed the onset of secondary cases in the family; the three patients whose families received "complete" prophylaxis developed the disease more than three months later. Successful eradication of carriage within the household cannot prevent outbreak strains re-entering the family; the interval depends on the prevalence and rate of transmission of outbreak strains in the local population.

Whether or not prophylaxis has been given the general practitioner and members of the family should remain vigilant after a case of meningococcal disease. A randomised controlled trial is needed to test the hypothesis that eradicating meningococcal carriage in household contacts prevents further cases of meningococcal disease.

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Nicotine absorption and dependence in an over the counter aid to stopping smoking

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The importance of nicotine dependence in cigarette smoking and as a deterrent to stopping is receiving increasing recognition.[1] This stems partly from the use of nicotine replacement methods to treat dependent smokers. Nicotine chewing gum, available only on prescription, is the only nicotine replacement treatment that is licensed in the United Kingdom. We investigated the absorption of nicotine from an over the counter aid to stopping smoking (Stoppers; Leo Laboratories).

Case report and study

A 38 year old man who had smoked hand rolled cigarettes for over 20 years stopped smoking with the help of nicotine chewing gum (Nicorette) 2 mg from his general practitioner. After he had used 15 pieces a day for two months he broke a tooth while chewing. He then started taking Stoppers, describing the transition as effortless, and was soon taking 30-60 lozenges a day. He contacted our clinic after a failed attempt to stop them after two years' use. Stopping taking Stoppers had resulted in his feeling irritable, ill at ease, unable to concentrate, depressed, and hungrier than usual. These symptoms of withdrawal from tobacco were rapidly relieved when he resumed taking Stoppers after four days' abstinence. We took a blood sample just after he had finished one lozenge, after a total of 20 on the day. Plasma nicotine and cotinine concentrations were 18·9 μg/l and 415 μg/l respectively. An expired air carbon monoxide concentration of 3 ppm confirmed that he had stopped smoking.

He bought his lozenges in bulk from the manufacturer, partly for economic reasons as a discount was offered and partly because of anxiety about running out. He also believed that these lozenges were stronger and more satisfying than lozenges purchased from pharmacists.

We tested lozenges obtained from local pharmacists and directly from the manufacturer. Four volunteers who no longer smoked took lozenges from both sources on a schedule of two every 30 minutes and allowed them to dissolve without sucking. Subjects were meant to take 28 lozenges over seven hours but some stopped before this because of nausea. Blood samples for analysis of nicotine concentrations were taken 30 minutes after the last dose. The mean plasma nicotine concentration achieved with supplies bought from a pharmacist was 14·6 μg/l after an average of 22 lozenges taken over five and a half hours. The mean concentration achieved with lozenges supplied by the factory was 22·3 μg/l after an average of 17 lozenges over four hours. The plasma nicotine concentration increased by a mean of 4·6 μg/l (range 3·6-5·2) over 30 minutes in three subjects who took two lozenges supplied by the factory.

Comment

Stoppers led to substantial absorption of nicotine. The concentrations from lozenges bought locally were higher than those from clinical use of 2 mg nicotine chewing gum,[2] whereas lozenges supplied by the factory gave concentrations similar to the lowest achieved from cigarette smoking[2] and to those achieved from chewing 4 mg gum on an imposed schedule.[3 4] Absorption from two lozenges was roughly similar to that from one piece of 2 mg gum.[5] The factory lozenges delivered more nicotine than those bought locally, confirming reports from patients and suggesting that the product may have a limited shelf life.

Our observations suggest that Stoppers have some therapeutic potential as a specific effect of nicotine in alleviating withdrawal from tobacco and promoting stopping smoking is now well established. The ease of taking the lozenges may make them suitable for dependent smokers who find chewing gum difficult or aversive. At the same time, there must be concern about the lack of information and guidance provided for the consumer and about the potential for abuse. The lozenges are not packaged in child proof containers, and the labelling does not mention nicotine, say why nicotine might be helpful, or point out any hazards of use.

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